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Computer Modeling of Acceleration Effects on Cerebral Oxygen Saturation

Richard A. McKinley
Lloyd D. Tripp, JR

Air Force Research Laboratory

Steve D. Bolia
Marvin A. Roark

General Dynamics
5200 Springfield Pike STE 200
Dayton OH 45431

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Human Effectiveness Directorate
Biosciences and Protection Division
Aircrew Performance and Protection Branch
Wright-Patterson AFB OH 45433-7947

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14. ABSTRACT
Introduction: Failure to effectively regulate BP and cerebral perfusion during high-G aircraft maneuvering may contribute to reduced performance in pilots due to the fact that perfusion to the peripheral cerebral tissues may not be adequate to support the mental demands of flight. Therefore, a critical area of investigation is the study of cortical tissue oxygenation responses to + Gz acceleration. Methods: Two experiments were used to build two sections of a cerebral oxygen saturation (rso2) model. Experiment 1: Six subjects participated in the study. A cerebral oximeter (gold standard) provided rso2. Acceleration profiles (subjects relaxed) included a 0.1 G .5-1 G onset to central light loss (CLL) and a 3 G .S-I onset to a G level that was 1 Gz above CLL to an endpoint of G-LOC. Experiment 2: There were 12 subjects (with G protection) who participated in this study. The rso2 data were collected during five different simulated aerial combat maneuvers. A model was created that read the Gz profile as input and calculated changes in rso2. The correlation coefficient, linear best-fit slope, and mean percent error were calculated to determine agreement. Results: The average value for the correlation coefficients, linear best-fit slopes, and mean percent errors for the unprotected subjects were 0.79, 0.87, and 6.08, respectively. These values for the protected subjects were 5 G (0.994, 1.011, 0.384), 6 G (0.994, 0.909, 0.811), 7 G (0.986, 1.061, 0.692), 8 G (0.969, 1.016, 1.300), and 9 G (0.994, .979, 0.558), respectively. Discussion: The model is a good predictor of rso2 values for protected and unprotected subjects under +Gz stress.

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RESEARCH ARTICLE

Computer Modeling of Acceleration Effects on Cerebral Oxygen Saturation

Richard A. McKinley, Lloyd D. Tripp, Jr., Steve D. Bolia, and Marvin R. Roark

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Introduction: Failure to effectively regulate BP and cerebral perfusion during high-G aircraft maneuvering may contribute to reduced performance in pilots due to the fact that perfusion to the peripheral cerebral tissues may not be adequate to support the mental demands of flight. Therefore, a critical area of investigation is the study of cortical tissue oxygenation responses to $+G_z$ acceleration. ***Methods:*** Two experiments were used to build two sections of a cerebral oxygen saturation (rSo_2) model. Experiment 1: Six subjects participated in the study. A cerebral oximeter (gold standard) provided rSo_2. Acceleration profiles (subjects relaxed) included a 0.1 $G \cdot s^{-1}$ G onset to central light loss (CLL) and a 3 $G \cdot s^{-1}$ onset to a G level that was 1 G_z above CLL to an endpoint of G-LOC. Experiment 2: There were 12 subjects (with G protection) who participated in this study. The rSo_2 data were collected during five different simulated aerial combat maneuvers. A model was created that read the G_z profile as input and calculated changes in rSo_2. The correlation coefficient, linear best-fit slope, and mean percent error were calculated to determine agreement. ***Results:*** The average value for the correlation coefficients, linear best-fit slopes, and mean percent errors for the unprotected subjects were 0.79, 0.87, and 6.08, respectively. These values for the protected subjects were 5 G (0.994, 1.011, 0.384), 6 G (0.994, 0.909, 0.811), 7 G (0.986, 1.061, 0.692), 8 G (0.969, 1.016, 1.300), and 9 G (0.994, 0.979, 0.558), respectively. ***Discussion:*** The model is a good predictor of rSo_2 values for protected and unprotected subjects under $+G_z$ stress.

Keywords: G physiology, blood flow, high G.

ACCELERATION-INDUCED cerebral perfusion insults have been a significant physiological threat to high-performance aircraft pilots since the development of the earliest fighter aircraft circa 1919. During tight turns and climbs, centripetal acceleration can be greatly increased. This is equated to multiples of the acceleration due to gravity (G). Highly maneuverable aircraft generate acceleration in the head-to-foot or z-axis.

The cerebral arterial tree can be approximated as a column of blood contained in non-distensible tubes. Therefore, increases in $+G_z$ acceleration cause an increase in weight or force (since weight is a measure of force due to gravity, i.e., mass multiplied by acceleration), which makes it much more difficult for the heart to pump blood through the carotid arteries into intracranial vessels. Each additional $+1\ G_z$ applied translates into 22 mmHg in eye-level BP (11). The flow of blood in the intracranial arteries significantly decreases once the hydrostatic pressure exceeds the ability of the cardiovascular system to generate compensating pressure changes, thus greatly inhibiting the amount of cerebral perfusion and oxygen saturation (2).

Because neurological activity requires a large amount of oxygen, it can be stated that cerebral metabolism will decrease with increased cerebral hypoxia. Consequently, information processing and cognitive function (i.e., the ability to make critical decisions) are seriously impaired (10). If the hypoxia is severe, neurological function will decrease to the point that only essential functions (such as breathing and heart contraction) will be maintained and loss of consciousness will occur. It is hypothesized that regional oxygen saturation (rSo_2) measurements can be correlated to cognitive performance decrements.

The theory behind cerebral oximetry is conceptually simple. Near infrared (NIR) light photons are transmitted into the skin over the forehead. After being scattered inside the skin, scalp, skull, and brain, some fraction of the injected photons survive to return and exit the skin. By measuring the quantity of returning photons as a function of wavelength, one can infer the spectral absorption of the underlying tissue and make some conclusions about its average oxygenation.

Previous work by others (12,13) indicates that at a source detector separation of 4 cm, some of the light injected by the near-infrared light emitting diode and received by NIR demonstrated that the NIR could penetrate the cranium and enter the cerebral cortex. Hongo et al. confirmed these results in the human forehead by injecting a bolus of infrared absorbing dye (indocyanine green) into the internal carotid artery and observing the transient decrease in signals at various source detector spacing (8).

The INVOS cerebral oximeter system (Somanetics, Troy, MI) was used to measure the cerebral oxygen saturation in the two experiments discussed in this

From the Aircrew Performance and Protection Branch, Air Force Research Laboratory, Wright-Patterson AFB, OH.

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Address reprint requests to: Richard A. McKinley, B.S., Aircrew Performance and Protection Branch, Air Force Research Laboratory, 2215 First St., Bldg. 33, Wright-Patterson AFB, OH 45433-7947; Richard.McKinley2@wpafb.af.mil.

paper. The oximeter is the gold standard in measuring oxygen saturation levels in the brain. It measures changes in rSo_2 within a sample of blood in the cerebral cortex. Sensors, consisting of a NIR light emitting diode juxtaposed to two NIR sensors, were placed on the left side of the subject's forehead and secured in place with an elastic bandage. These sensors were then connected to the oximeter and the monitor. Detailed working principles and validation of the cerebral oximeter were well described by Henson et al. and Hongo et al. (7,8).

The primary focus of this effort was to build a model that could effectively and reliably predict changes in rSo_2 within the cerebrum due to $+G_z$ loading. It was desirable to design the model in such a way that it could make accurate predictions despite variations in the G_z profile. This paper reports the resulting findings.

METHODS

Modeling efforts were separated into two sections; one to calculate cerebral oxygen saturation ($\%rSo_2$) changes in the relaxed, unprotected individual, and the other to predict $\%rSo_2$ changes in the straining, protected subject. Likewise, two separate experiments were used to build each section of the model. Each experiment used a completely separate set of subjects and each subject was a member of the Air Force Research Laboratory Sustained Acceleration Panel. However, there was no overlap of the subject populations. All participants were required to meet Air Force Flying Class III medical standards and to have no history of neurological pathology or of having experienced episodes of loss of consciousness. Both experiments were conducted at the Air Force Research Laboratory's centrifuge facilities located at Wright-Patterson AFB, OH, and were approved by the base's Institutional Review Board. The gondola of the centrifuge was equipped with an F-16-like ACES II ejection seat with a seatback reclined to 30° from vertical. The seat was modified with adjustable head and shoulder supports that prevented the participant's head and torso from sliding off the seat during a G-LOC episode. The cerebral oximeter was installed inside the gondola and was used to measure rSo_2 within the cerebrum for each of the two experiments.

A two-way communication system provided voice-communication between the research participant and the investigator. The participant's microphone was fixed in the open position to allow the participant "hands-free" communication. Participants were also provided with an emergency abort switch that enabled them to stop the centrifuge at any time during testing. Each subject completed an informed consent form approved by an Institutional Review Board from Wright-Patterson Air Force Base.

Experiment 1

Six subjects (three women and three men) volunteered to participate in the study. Participants wore a standard Air Force issue Nomex® flight suit and a Gentex (Carbondale, PA) HGU-55/P flight helmet during all testing runs. A computer control system was used to generate two acceleration profiles. The first was a gradual G-onset rate (GOR) of 0.1 $G \cdot s^{-1}$. This profile was employed to establish the participant's relaxed G-tolerance level (the G level at which eye-level BP can no longer be maintained) on a given test day. As the centrifuge was slowly accelerated, participants were asked to view the red central target that represented 10° of the visual field. Subjects were instructed to execute an anti-G straining maneuver (AGSM) when the visual field blackened out and all that could be seen was the target, i.e., central light loss or CLL occurred. The G exposure was aborted when the subject initiated an AGSM. The G level at which this occurred was termed GOR_{max}.

The second G profile, which was run after the determination of GOR_{max}, featured a rapid G-onset rate (ROR) of 3 $G \cdot s^{-1}$ to a pre-established target level that was set individually for each participant on each testing day. The target level was determined by adding +1 G_z to the GOR_{max}. The principal investigator terminated the ROR profile when G-LOC occurred or the G profile reached a time limit of 15 s. The presence of G-LOC was determined subjectively using the following criteria that included the following signs: 1) slumping of the head and upper body; 2) dual eye closure; and 3) jaw muscle relaxation reflected in a gaping mouth (14). All three signs needed to be present in order to determine that a participant had gone into G-LOC and the principal investigator and the flight surgeon had to be in total agreement in order to make the call.

Experiment 2

There were 12 subjects (6 men, 6 women) who volunteered to participate in the study. Each subject was given a helmet and mask and fitted for a Combined Advanced Technology Enhanced Design G-Ensemble (COMBAT EDGE) with a standard CSU 13-BP anti-G suit. A computer control system was used to generate five different acceleration profiles. Each profile was a simulated aerial combat maneuver (SACM) and differed only in peak G_z (either 5, 6, 7, 8, or 9 G_z) achieved and overall duration. Each SACM contained two 5-s plateaus at the peak G_z value and various lower G plateaus. The SACMs all started from a baseline of 1.5 G_z and proceeded to a 3-G plateau that lasted approximately 10 s. This was followed by the first 5-s plateau at peak G_z, which was in turn followed (in order) by a 4.5, 3.0, 5.0, and 4.0 G_z plateau (each lasting 5 s). These G_z levels were followed by the second peak to the maximum G_z level for the particular SACM. Each profile ended with another 5-s plateau at 3 and then 6 G_z (5 G_z for the 5-G SACM) before returning to the baseline G_z level.

On a typical experimental day, subjects donned a standard G-suit and the corresponding COMBAT EDGE system (with helmet and mask). A brief medical evaluation was conducted by the medical personnel prior to the subject's insertion into the gondola of the centrifuge. Once securely fastened in the centrifuge, the subject performed four repetitions of a SACM profile, starting with the 5-G SACM on the first day. On each subsequent day, the peak G_z of the SACM profile would be increased by one. For example, on day 1 the subjects

performed the 5-G SACM, day 2 they performed the 6-G SACM, day 3 they performed the 7-G SACM, etc. Subjects were instructed to execute an AGSM as needed to retain clear vision throughout each G exposure. A 2-min rest period was administered between each profile repetition on each experimental day.

Model

A computer-based model of cerebral saturation variations due to $+G_z$ stress was created using Microsoft Visual C++ 6.0. Due to the fact that human physiology is significantly different for subjects with G protection and those without, the model was broken into two separate sections. The first modeled subjects that were relaxed and did not have the benefit of a G-suit. The second section modeled subjects that were using the AGSM and a standard G-suit with COMBAT EDGE. The time history of the G_z profiles for each trial were recorded and then used as the input for both sections of the model. At the start-up window for either section of the program, the investigator was prompted to input the number of data points in the G_z profile and the mean percent cerebral oxygen saturation baseline value for the particular subject and trial. The software then employed the G_z and time values to calculate changes in rSo_2.

The first segment of the model, designed for relaxed subjects without G protection, used the single peak G_z profiles with a peak that was +1 G_z above GOR_{max}. To account for the physiologic time delay between the onset of G_z and the initialization of rSo_2 decrements, the model postponed significant changes in cerebral saturation for approximately 7–10 s following a considerable (> 0.3 G_z from baseline value) increase in G_z. The rate at which the rSo_2 declined was based on a percentage of the subject's baseline, the duration of the G peak, and the magnitude of the G peak. The rSo_2 decrement variable was set to a percentage of the subject's baseline and was subtracted from the previous value of rSo_2 at a frequency of 0.4 Hz. This percentage is variable based on peak G ranges (i.e., for higher G levels, the software uses a higher percentage of the subject's baseline rSo_2 to calculate the rate of decrease in rSo_2). For the G_z profiles used in this portion of the model (peak G_z from 5.9–7.6 G_z), it was optimal to use 9% of the baseline as the decrement variable for the rSo_2. Thus, for a subject with a baseline rSo_2 value of 70, the software would predict a decrease of 6.3 in rSo_2 for the first data point (2.5 s) following a significant increase in G_z. The rSo_2 decrement variable was also attenuated by 35% for each subsequent data point until the G_z level returned to a value that was less than or equal to half of the peak value. At this point, rSo_2 recovery was initiated using an increase of $1.832\ rSo_2 \cdot s^{-1}$. The recovery was terminated once rSo_2 values returned to baseline. Initial model development was completed using data from one of the six subjects. Data from the remaining five subjects were then used to determine the overall validity.

The second portion of the model, designed for subjects wearing the COMBAT EDGE system, a standard G-suit, and performing the AGSM, was constructed using the 5-, 6-, 7-, 8-, and 9-G SACM profile from experiment two. This section of the model also postponed significant decreases in rSo_2 by 7–10 s to account for the physiologic time delay. Due to the fact that G protection (AGSM, standard G-suit, and COMBAT EDGE) helps keep oxygenated blood in the cortical areas of the brain for longer periods of time, the decreases in rSo_2 are not as severe as they would be for an unprotected subject.

To allow for this difference, the initial decrease in rSo_2 was set to 0.9% of the subject's baseline. This initial decrement value was subtracted from the previous value of rSo_2 each time the G_z data was updated (every 2.5 s). However, this decrement value was variable and was modified by a factor that was set according to the magnitude of the most recent G peak achieved in the profile. Therefore, rSo_2 decreased more rapidly at higher G_z and more slowly at lower G_z. Recovery was initialized under two conditions. The first condition was that the G_z level returned to a value that was 26% of its peak value. The second condition was that the G_z had achieved a value that was less than 1.7 G_z. The recovery was a linear increase with a slope equal to the last value of the decrement variable multiplied by 2.276.

The time history of measured rSo_2 was compared with the predicted values produced by the model. For the first segment of the model, the determination of the agreement focused on the acute rSo_2 decrease event. The second portion of the model used the entire SACM profile to determine overall agreement, or closeness of fit. Agreement was defined by criteria established by Griffin, who maintains that agreement requires a high positive correlation (close to 1), a slope of unity (on a plot of measured vs. predicted values), and low error (i.e., the difference between measured and predicted values is low) (6). Therefore, for the first segment of the model, the correlation coefficient, slope (calculated on a plot of measured vs. predicted values), and mean percent error were calculated for each trial. The mean was then calculated for each of the three values.

The first segment of the model also generated an average rSo_2 profile using the mean rSo_2 baseline value and the average G_z profile. The average rSo_2 values were normalized across subjects by transforming the rSo_2 data to represent a percent change from baseline. The two profiles were then compared using the same calculations and agreement criteria stated previously.

The second portion of the model generated an rSo_2 profile for each of the different SACMs. These were the same across repetitions due to the fact that the SACMs did not change. These profiles were converted values that represent the percent change from baseline. The raw collected data were then averaged across subjects and repetitions and compared with the profiles predicted by the model using the same calculations and agreement criteria stated previously.

RESULTS

Subject and model data were analyzed using standard agreement calculations reported by Griffin (2001) (6). The first subunit of the model was verified with data from G-LOC research where each G profile con-

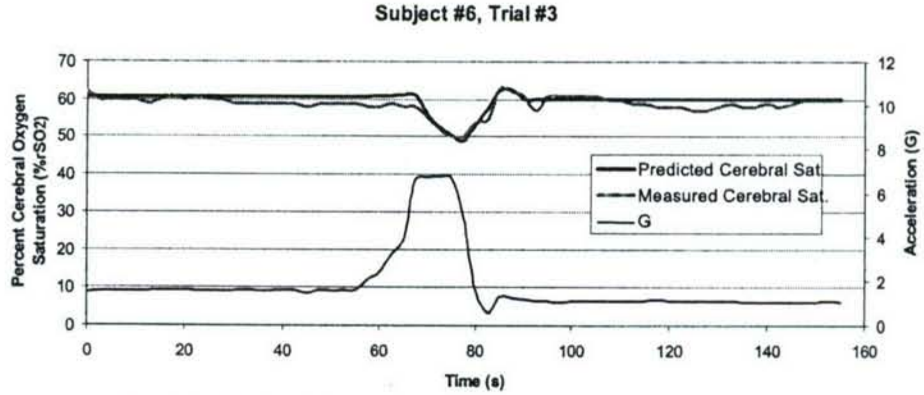

Fig. 1. Example plot of measured vs. predicted rSo_2 values (highest agreement).

tained a single peak between 5.9 and 7.6 G. The averages and standard deviations for the correlation coefficients, the slopes, and the error are 0.790 ± 0.166, 0.869 ± 0.342, and 6.079 ± 2.412, respectively.

For the first model segment, measured vs. predicted rSo_2 values were plotted for each G_z data run. **Fig. 1** presents the trial with the highest agreement, whereas **Fig. 2** displays the trial with the lowest agreement. The first segment of the model also generated an average rSo_2 profile (based on an average G_z profile and an average rSo_2 baseline value) that was compared with the average profile for the measured rSo_2 data. The two were then compared using the previous agreement criteria. The correlation coefficient, slope, and mean percent error were 0.885, 0.6549, and 2.14, respectively.

The second subunit of the model was verified using data from subjects performing 5-, 6-, 7-, 8-, and 9-G SACMs. The value for the correlation coefficients, linear best-fit slopes, and mean percent errors were: 5 G, 0.994, 1.011, 0.384; 6 G, 0.994, 0.909, 0.811; 7 G, 0.986, 1.061, 0.692; 8 G, 0.969, 1.016, 1.300; and 9 G, 0.994, 0.979, 0.558; respectively. Each type of SACM was plotted with the rSo_2 model prediction profile and the average measured rSo_2 profile. The data was transformed into values that represent the percent change in rSo_2 from the baseline value. An example plot is shown in **Fig. 3**.

DISCUSSION

Modern high performance fighter aircraft are capable of achieving extremely high acceleration during turning and climbing maneuvers that can typically apply up to $+9$ G_z on the pilot. Accelerations of this magnitude and direction are sufficient to drive a sudden and rapid drop in cerebral BP and, thus, cerebral oxygen saturation. A cerebral perfusion failure during high-G aircraft maneuvering is likely to contribute to reduced performance in pilots and may be attributed to decreased perfusion to the peripheral cerebral tissue. Research has indicated that observers undergoing hypoxia are subject to mental blocks, a general slowing of early information processing, and a deterioration of sensory functions (dark adaptation), cognitive functions (concentration, verbal and visual memory, target acquisition), and motor skills (tracking, complex psychomotor performance) (1,3–5,9,10). In extreme cases, G-induced cerebral hypoxia can result in a loss of consciousness.

The ability to model and predict rSo_2 decrements due to G_z is important for several reasons. First, this modeling effort may prove to be useful when planning air combat missions, sorties, or training exercises. The model would be capable of predicting the extent of rSo_2 decrements based on an estimated G_z profile given possible combat threats and aircraft capabilities. An-

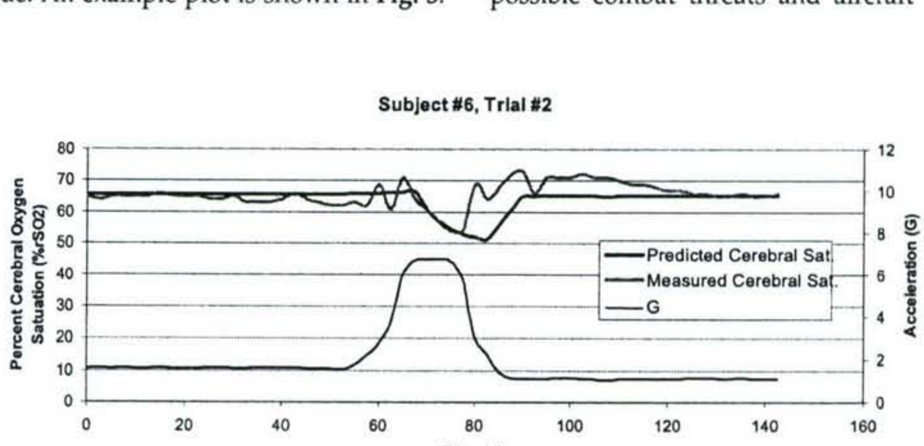

Fig. 2. Example plot of measured vs. predicted rSo_2 values (lowest agreement).

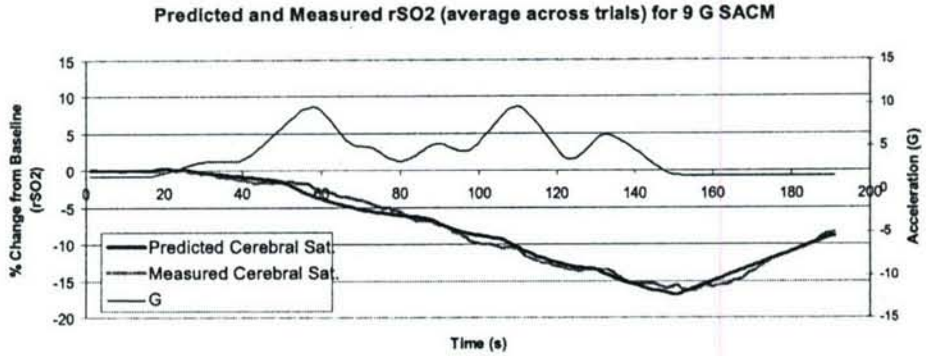

Fig. 3. Example plot of measured vs. predicted rSo_2 values (9-G SACM).

other application of this model may lie in training and exercise planning. Instructors would be able to identify G_z profiles that presented an elevated level of danger for students on an individual basis. Furthermore, instructors would theoretically be able to choose aircraft maneuvers for students that would generate the appropriate G_z levels to decrease rSo_2 values to various degrees for safer demonstrations of decreased cognitive function that may occur in combat.

The second possible application resides in flight simulation. The model could easily be integrated into existing pilot training simulator programs. The second segment of the model encompasses the full range of G_z values (namely 1.0–9.0 G_z) that high performance fighter pilots experience in flight and has been verified for long-duration, multiple peak profiles that resemble the G_z levels pilots might experience during air combat maneuvers. It also provides evidence of the fact that rSo_2 does not recover, even at G_z levels between 3 and 5, until the G_z has been substantially decreased (usually less than 2.5 G_z), which could be a very important detail when modeling pilot behavior during air combat. The model could predict rSo_2 decreases for computer-controlled adversaries during standard combat maneuvering based on G_z level and time. Through relating percent decrements in rSo_2 to task performance, the simulated pilots could potentially experience performance deficiencies (and even loss of consciousness) due to $+G_z$ loading. This would offer a more robust simulation that reacts realistically with the dynamic environment.

Although the model was found to have good agreement with the data in this experiment, it has several limitations. Currently, the first section of the model has only been analyzed for a relatively narrow range of G_z peak values (5.9–7.6 G_z). Therefore, before the model of the unprotected subjects could be fully implemented, it would need to be compared with rSo_2 values over the entire range of peak G_z values (namely 1.0–9.0 G_z) that high performance fighter pilots experience in flight. Furthermore, the rSo_2 values have not yet been fully related to cognitive and performance deficiencies based on the level of hypoxia. It light of this fact, it would be difficult to correlate a lack of cerebral perfusion to a percentage decrement in simulated task performance. However, both model segments yield an important first step in building a larger model that would be useful for a wider range of applications.

The results of this study initially revealed a high correlation between the measured rSo_2 data and the predicted rSo_2 values. However, a positive correlation between values measured and those predicted by a model is not sufficient to validate the model (6). For this reason, slope (on a plot of measured vs. predicted values) and mean percent error (calculated on a plot of measured vs. predicted values) were calculated in addition to the correlation coefficients for each data run. The results revealed exceptional agreement between the model predictions and measured data due to the fact that the mean correlation coefficient was effectively close to one, the mean slope was close to one, and there was relatively low error. This analysis reinforces the conclusion that the model is a valid predictor of rSo_2 values for both G_z profiles with peaks between 5.9 and 7.6 G_z (for unprotected subjects), as well as long-duration (up to 190 s), variable peak G_z (between 1.0 and 9.0) profiles (for protected subjects).

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